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Actin Modifications and Calcium Homoeostasis in Neurotoxicity. The Case of Organotin Salts

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Abstract

The cytoskeleton is a major constituent of the neuronal cytoplasm; it controls cell shape and plays important roles in regulating various physiological processes. In neurons, actin filaments are involved in the growth of the neurite and the neurotransmitter release. Recent findings suggest that actin filaments play a role in modulating $[Ca^{2+}]_i$ responses to neurotoxic insults. The physiological functioning of the neural cell is critically dependent on the intracellular distribution of calcium. An increase of cytosolic free calcium can activate a number of intracellular reactions, including neurotransmitter release, protein phosphorylation, protease activity, and, eventually, cell death. Many neurotoxic agents with diverse mechanisms have been reported to affect mechanisms associated with calcium. Among these are organotin compounds: they can both raise the cytosolic and synaptosomal $[Ca^{2+}]_i$ concentrations and interfere with the $[Ca^{2+}]_i$ response evoked by different agonists. Furthermore, some of these compounds cause actin depolymerization. The interference of triethyltin (TET)--a compound inducing myelin vacuolization and brain oedema--with Ca^{2+} homoeostasis and actin polymerization results in an adverse effect on neurotransmitter release in different neural cell lines. However, another neurotoxic organotin compound (trimethyltin, TMT) induces apoptosis in neural cells through the activation of a Ca^{2+} -dependent pathway. In conclusion, the identification of the key changes in actin and Ca^{2+} homoeostasis could give early information on neural cell perturbation resulting in altered functionality or even cell death.

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Original article

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Abstract

Glaucoma is a leading cause of blindness worldwide and the second leading cause of irreversible blindness in the USA. The most common form of glaucoma, primary open angle glaucoma, is characterized by a chronically elevated intraocular pressure in the absence of any demonstrable structural abnormalities in the eye. The pathologic hallmark of glaucomatous optic neuropathy is the selective death of retinal ganglion cells associated with structural changes in the optic nerve head. Recent discoveries suggest a role for nitric oxide, glutamate, apoptosis, and others, in the pathophysiology of this neuropathy. These newer discoveries are addressed in this article.

Author Keywords: apoptosis; caspase; glaucoma; glutamate; nitric oxide; retinal; ganglion cells



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